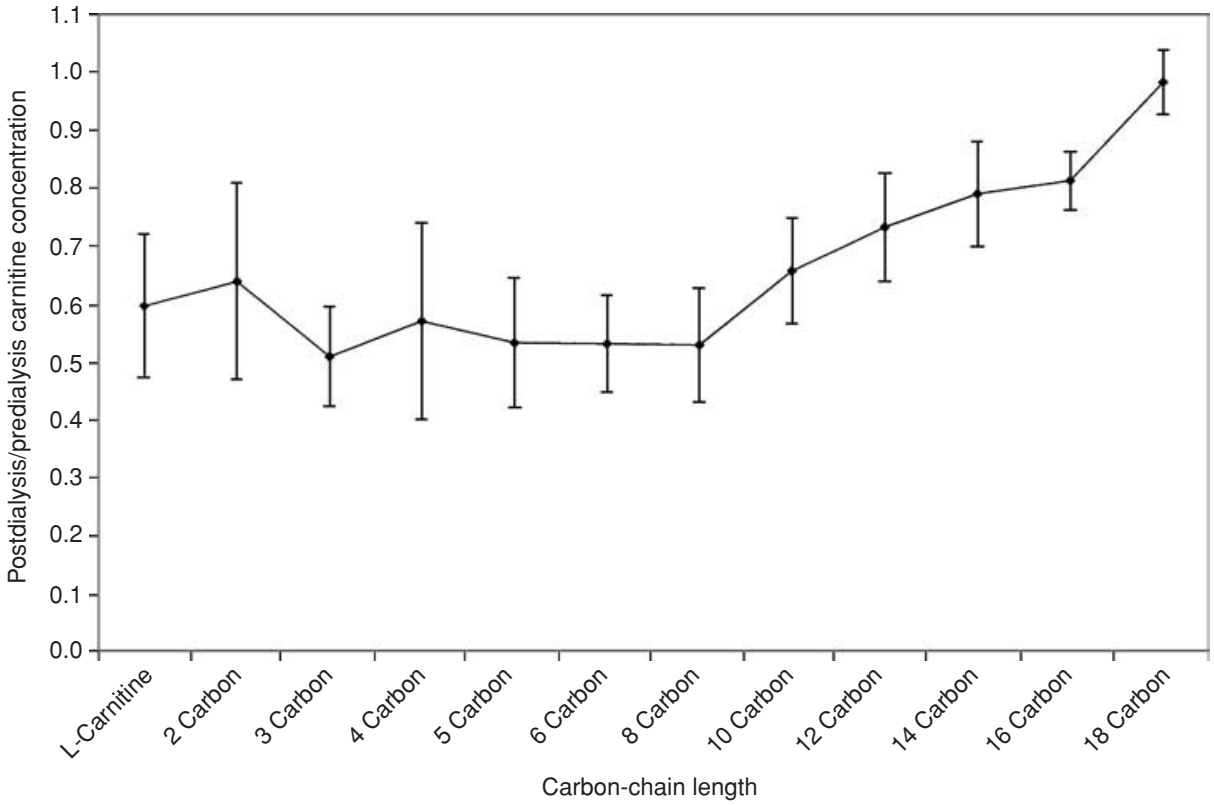

Fig. 1. Relationship between mean (± SEM) ratio of postdialysis/predialysis acylcarnitine concentrations and carbon-chain length of the acyl group.

significant correlation between carbon-chain length and dialytic removal (Fig. 1).

In light of these findings, we propose that, in combination with disturbed metabolism, the observed alterations in the carnitine pool are contributed to by a decrease in the dialytic removal of acylcarnitines, in particular long-chain acylcarnitines, most likely a result of the increased molecular weight and lipophilicity that accompanies increased chain length.

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Albuminuria in patients with hepatitis C: Is inflammation the missing link?

To the Editor: The study by Liangpunsakul and Chalasani on the association between hepatitis C and microalbuminuria reports an independent association between these 2 clinical entities [1]. The significance of their results may, however, be limited by the fact that their use of 1 albumin to creatinine ratio to measure microalbuminuria may be a source of error, including an underestimation of microalbuminuria in some some racial/ethnic groups [2].

Futhermore, their failure to find an association between hepatits C and the metabolic syndrome, may be explained by the fact that in at least in some age groups,

microalbuminuria may not be necessarily a component of the metabolic syndrome [3].

Finally, on the basis of other studies, one may speculate that chronic inflammation may be the underlying mechanism that explains the association of hepatic C with microalbuminuria because atherosclerosis itself appears to be an inflammatory disorder [4, 5].

In their study, markers of inflammation such as IL-6 and C-reactive protein were not measured.

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Reply from the Authors

We are very grateful to Dr. Nzerue for his thoughtful comments on our paper [1]. In this correspondence, we respond to thoughtful queries by Dr. Nzerue.

First, in our discussion, we admitted that a single measurement of urine albumin to creatinine ratio to assess for the presence of microalbuminuria is not ideal. Unfortunately, the NHANES III is a cross-sectional study and sequential urinary data were not available.

Second, Dr. Nzerue points out that there is not a consistent relationship between the metabolic syndrome and microalbuminuria in some age groups, and this may explain why we failed to observe an association between hepatitis C and the metabolic syndrome. While this is plausible, more rigorous studies are needed to examine this issue as our results disagree with a previous retrospective cohort study [2], which showed a high prevalence of the metabolic syndrome in patients with hepatitis C.

Third, we accept the possibility that chronic inflammation or immune processes are involved in the pathogenesis of microalbuminuria in hepatitis C subjects. We explored this possibility, but found that cryoglobulin levels were not measured in the NHANES III, and rheumatoid factor data were missing in most patients in the hepatitis C cohort. However, we failed to find any differences in the levels of C-reactive protein in subjects with hepatitis C and controls (0.41 ± 0.62 vs. 0.44 ± 0.86 mg/dL, $P = 0.5$).

Our study should be viewed as exploratory in nature and our observations require validation in ongoing prospective studies (e.g., NIDDK-funded Virahep-C study).

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Red blood cell deformability and diabetic nephropathy

To the editor: In an interesting study, Brown et al [1] found an impaired red blood cell deformability in 57 adult type 2 diabetic patients. They used a filtration technique using polycarbonate membranes with straight channels of 3 micrometer pore diameter. In patients with diabetic nephropathy they found an increased impairment in red blood cell deformability. The hypothesis that an impaired red blood cell deformability contributes to renal function decline is very attractive and may give rise to therapeutic possibilities. Many studies found an impaired red blood cell deformability in diabetic patients; others, however, did not. Years ago we tested the hypothesis that red blood cell deformability contributes to diabetic nephropathy using both ektacytometry and erythrocyte filtration, with micropores with a diameter of 5 micrometer [2]. In order to imitate local circumstances in the kidney red blood cell, deformability was also measured in hyperosmolar solutions. Seventy-one insulin-dependent diabetic patients were included, 25 patients without any sign of organ damage, 21 patients with microalbuminuria, 13 patients with overt nephropathy, and 12 patients with leg ulceration. No decreased red blood cell deformability was found in any of the diabetic groups with either technique, neither